L Number	Hits	Search Text	DB	Time stamp
1	0	ulcer$ same ("IL-1A -889" or "IL1A-889" or "Il-1A-889" or (IL-1A near3 "-889"))	USPAT; US-PGPUB; DERWENT	2002/06/25 16:33
2	0	(IBD or "Chrohn's Disease") same ("IL-1A -889" or "IL1A-889" or "Il-1A-889" or (IL-1A near3 "-889"))	USPAT; US-PGPUB; DERWENT	2002/06/25 16:35
3	1	(IBD or "Chrohn's Disease" or ulcer$) same ("IL-1b +3953" or "IL1b+3953" or "Il-1b+3953" or (IL-1B near3 "+3953"))	USPAT; US-PGPUB; DERWENT	2002/06/25 16:36

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     (FILE 'HOME' ENTERED AT 16:01:18 ON 21 JUN 2002)

     FILE 'MEDLINE, CAPLUS' ENTERED AT 16:01:24 ON 21 JUN 2002
L1          101 S (IL-1A OR IL-1B OR IL-1R OR IL-6 OR IL-8 OR TNFA OR TNF.ALPHA
L2           64 DUP REM L1 (37 DUPLICATES REMOVED)

     FILE 'STNGUIDE' ENTERED AT 16:20:12 ON 21 JUN 2002

     FILE 'MEDLINE, CAPLUS' ENTERED AT 16:31:10 ON 21 JUN 2002
L3            1 S (IL-1A OR IL-1B OR IL-1R OR IL-6 OR IL-8 OR TNFA OR TNF.ALPHA
L4            1 S (IL-1A OR IL-1B OR IL-1R OR IL-6 OR IL-8 OR TNFA OR TNF.ALPHA
L5           30 S (IL-1A (2A) "-889")  OR (IL-1B (2A) "+3953")
L6           21 DUP REM L5 (9 DUPLICATES REMOVED)
L7            1 S L6 AND ULCER?
L8          986 S DERMAL ULCER? OR VENOUS ULCER? OR DECUBITIS ULCER?
L9          934 DUP REM L8 (52 DUPLICATES REMOVED)
L10           0 S L9 AND (POLYMORPHISM? OR MUTATION?) AND (CYTOCKINE?)
L11           1 S L9 AND (POLYMORPHISM? OR MUTATION?) AND (CYTOKINE?)
L12           1 S L9 AND (POLYMORPHISM? OR MUTATION?) AND (INFLAMM?)
L13           1 S L9 AND (POLYMORPHISM? OR MUTATION?) AND (INFLAM?)
L14         263 S NESTED PCR AND IMPROVE?
L15         182 DUP REM L14 (81 DUPLICATES REMOVED)
L16         394 S PROBE? (10A) (LABEL? ) (10A) (CHEMILUMIN?)
L17         299 DUP REM L16 (95 DUPLICATES REMOVED)

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L1 101 (IL-1A OR IL-1B OR IL-1R OR IL-6 OR IL-8 OR TNFA OR TNF.ALPHA. OR INTERLEUKIN 1 OR INTERLEUKIN 6 OR INTERLEUKIN 8) (P) (MUTATIO N? OR POLYMORPHISM?) (P) (ULCER? OR IBD OR INFLAMMATORY BOWEL DISEASE)

1 (IL-1A OR IL-1B OR IL-1R OR IL-6 OR IL-8 OR TNFA OR TNF.ALPHA. OR INTERLEUKIN 1 OR INTERLEUKIN 6 OR INTERLEUKIN 8) (P) (MUTATION? OR POLYMORPHISM?) (P) (CHRONIC ULCER? OR DERMAL ULCER? OR VENOUS ULCER OR PRESSURE SORES OR DECUBITIS ULCER?)

L4 1 (IL-1A OR IL-1B OR IL-1R OR IL-6 OR IL-8 OR TNFA OR TNF.ALPHA. OR INTERLEUKIN 1 OR INTERLEUKIN 6 OR INTERLEUKIN 8) AND (MUTATIO N? OR POLYMORPHISM?) AND (CHRONIC ULCER? OR DERMAL ULCER? OR VENOUS ULCER OR PRESSURE SORES OR DECUBITIS ULCER?)

L2 ANSWER 63 OF 64 MEDLINE
AN 94164479 MEDLINE
DN 94164479 PubMed ID: 8119534
TI Novel genetic association between ulcerative colitis and the anti-inflammatory cytokine interleukin-1 receptor antagonist.
AU Mansfield J C; Holden H; Tarlow J K; Di Giovine F S; McDowell T L; Wilson A G; Holdsworth C D; Duff G W
CS Department of Medicine and Pharmacology, University of Sheffield, England.
SO GASTROENTEROLOGY, (1994 Mar) 106 (3) 637-42.
Journal code: 0374630. ISSN: 0016-5085.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199404
ED Entered STN: 19940412
Last Updated on STN: 19940412
Entered Medline: 19940401
AB BACKGROUND/AIMS: **Ulcerative** colitis and Crohn's disease have well-recognized familial tendencies, but the genetic basis of this clinical observation remains unknown. The cytokine **interleukin-1** receptor antagonist is a potent anti-inflammatory protein that can prevent immune-mediated bowel inflammation in animals. We have previously characterized a **polymorphism** within the gene for this cytokine and others in the genes for the proinflammatory cytokines **interleukin 1** alpha, **interleukin 1** beta, and tumor necrosis factor alpha. The aim of this study was to determine whether **inflammatory bowel disease** was associated with particular alleles of these polymorphic cytokine genes. METHODS: The allelic frequencies of these polymorphic cytokine genes were determined in patients with **ulcerative** colitis (n = 113), Crohn's disease (n = 78), and healthy controls (n = 261). RESULTS: Allele 2 of **interleukin-1** receptor antagonist was significantly over-represented in the **ulcerative** colitis patients: 35% versus 24% in controls (P = 0.007). Carriage of at least one copy of this allele gave an odds ratio of 2.0 for **ulcerative** colitis compared with controls. This association with allele 2 of **interleukin 1** receptor antagonist was greatest in patients with total colitis and was not seen in Crohn's disease. There were no associations between UC and any of the other cytokine genes examined. CONCLUSIONS: This observation provides evidence that **interleukin-1** receptor antagonist may have a role in determining the genetic susceptibility to and pathogenesis of **ulcerative** colitis.

L2 ANSWER 62 OF 64 CAPLUS COPYRIGHT 2002 ACS
AN 1996:1268 CAPLUS
DN 124:53397
TI Allelic polymorphism in IL-1.beta. and IL-1 receptor antagonist (IL-1Ra) genes in inflammatory bowel disease
AU Bioque, G.; Crusius, J. B. A.; Koutroubakis, I.; Bouma, G.; Kostense, P. J.; Meuwissen, S. G. M.; Pena, A. S.
CS Departments Gastroenterology, Free University Hospital Amsterdam, Amsterdam, Neth.
SO Clin. Exp. Immunol. (1995), 102(2), 379-83
CODEN: CEXIAL; ISSN: 0009-9104
DT Journal
LA English
AB Recent reports have shown that allele 2 of the IL-1 receptor antagonist (IL-1Ra) gene is overrepresented in ulcerative colitis (UC). Healthy individuals carrying allele 2 of this gene have increased prodn. of IL-1Ra protein. Since the final outcome of the biol. effects of IL-1.beta. may depend on the relative proportion of these two cytokines, the authors have studied if a TaqI polymorphism in the IL-1.beta. gene, which is relevant to IL-1.beta. protein prodn., may be involved in the genetic susceptibility to UC and Crohn's disease (CD), in assocn. with the established IL-1Ra gene polymorphism. Polymorphisms in the closely linked genes for IL-1.beta. and IL-1Ra were typed in 100 unrelated Dutch patients with UC, 79 with CD, and 71 healthy controls. The polymorphic regions in exon 5 of the IL-1.beta. gene and in intron 2 of the IL-1Ra gene, were studied by polymerase chain reaction (PCR)-based methods. The IL-1.beta. allele frequencies in UC and CD patients did not differ from those in healthy controls. To study if the IL-1.beta. gene polymorphism might participate synergistically with the IL-1Ra gene polymorphism in susceptibility to UC and CD, individuals were distributed into carriers and non-carriers of allele 2 of the genes encoding IL-1.beta. and IL-1Ra, in each of the patient groups and controls. Results indicated a significant assocn. of this pair of genes, estd. by the odds ratio (OR) after performing Fisher's exact test, in the UC group ($P = 0.023$, $OR = 2.81$), as well as in the CD group ($P = 0.01$, $OR = 3.79$). Thus, non-carriers of IL-1.beta. allele 2 were more often present in the subgroup of patients carrying the IL-1Ra allele 2. By contrast, no assocn. of these alleles was detected in the group of healthy controls ($P = 1.00$, $OR = 0.92$). These results suggest that the IL-1.beta./IL-1Ra allelic cluster may participate in defining the biol. basis of predisposition to chronic inflammatory bowel diseases

L2 ANSWER 56 OF 64 MEDLINE DUPLICATE 33
AN 96188949 MEDLINE
DN 96188949 PubMed ID: 8608636
TI Distribution of four polymorphisms in the tumour necrosis factor (TNF) genes in patients with inflammatory bowel disease (IBD).
AU Bouma G; Xia B; Crusius J B; Bioque G; Koutroubakis I; Von Blomberg B M; Meuwissen S G; Pena A S
CS Department of Gastroenterology, Free University Hospital, Amsterdam, The Netherlands.
SO CLINICAL AND EXPERIMENTAL IMMUNOLOGY, (1996 Mar) 103 (3) 391-6. Journal code: 0057202. ISSN: 0009-9104.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199605
ED Entered STN: 19960605
Last Updated on STN: 19960605
Entered Medline: 19960528
AB In 153 patients with **IBD**, 64 with Crohn's disease (CD), and 89 with **ulcerative** colitis (UC), as well as in 54 healthy controls (HC), the frequencies of four known di-allelic **polymorphisms** in the genes for **TNF-alpha** and lymphotoxin alpha (LTalpha) were investigated. In the Dutch population, the alleles of these four **polymorphisms** are present in only five combinations, called TNF haplotypes: TNF-C, -E, -H, -I, -P. Furthermore, the relation with the presence of perinuclear anti-neutrophil cytoplasmic autoantibodies (P-ANCA) was studied. A small, but statistically significant, association between the **polymorphism** at position -308 in the promoter region of the **TNF-alpha** gene and UC was found. The frequency of the uncommon **TNF-alpha** -308 allele 2 was found to be decreased in patients with UC compared with HC (allele frequency of allele 2 in UC patients 0-15 versus 0.25 in HC, P=0.044). No significant differences in distribution of the TNF haplotypes were found between **IBD** patients and HC, although there was a tendency towards a higher frequency of the TNF-C haplotype in UC patients compared with controls (haplotype frequency 22% versus 13%; P=0.19). No statistically significant differences in distribution of the TNF haplotypes were observed between P-ANCA-positive and P-ANCA-negative UC patients. The strength of the associations indicates that TNF genes are not markers for the predisposition to suffer from **IBD**. They may, however, be markers of subsets of patients with UC and CD.

L2 ANSWER 50 OF 64 MEDLINE
AN 97347874 MEDLINE
DN 97347874 PubMed ID: 9203941
TI Lack of association between an **interleukin-1** receptor antagonist gene **polymorphism** and **ulcerative** colitis.
AU Hacker U T; Gomolka M; Keller E; Eigler A; Folwaczny C; Fricke H; Albert E; Loeschke K; Endres S
CS Medizinische Klinik, Klinikum Innenstadt, University Munich, Germany.
SO GUT, (1997 May) 40 (5) 623-7.
Journal code: 2985108R. ISSN: 0017-5749.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199707
ED Entered STN: 19970721
Last Updated on STN: 19970721
Entered Medline: 19970710
AB BACKGROUND: Recently, the association of a **polymorphism** in the gene coding for the anti-inflammatory cytokine **interleukin-1** receptor antagonist with **ulcerative** colitis has been reported. This was interpreted as a possible genetic predisposition for severity of the inflammatory response. AIMS: To examine this **polymorphism** in a southern German population. SUBJECTS: The study included 234 healthy controls, 57 patients with **ulcerative** colitis, including 31 patients with pancolitis, 44 first degree healthy relatives of patients with **ulcerative** colitis, and 65 patients with Crohn's disease. METHODS: Genotypes were determined by a polymerase chain reaction amplification of the intron 2 fragment harbouring a variable number of tandem repeat nucleotide sequences. Amplification products were separated on a 2% agarose gel. RESULTS: The allele frequency for allele 2 was 27% in healthy controls, 28% in Crohn's disease, and 21% in patients with **ulcerative** colitis. The same allele frequency (21%) was found in a subgroup of patients with **ulcerative** colitis affecting the whole colon. Thus for allele 2 as well as for all other alleles, genotypes, or carriage rates no significant differences were found compared with controls. All allele frequencies in the control population were similar to those in earlier studies. CONCLUSIONS: No association of a **polymorphism** in the **interleukin-1** receptor antagonist gene with **ulcerative** colitis could be identified in this southern German population. The findings of an earlier study reporting an increased frequency of allele 2, particularly in patients with pancolitis, could not be confirmed.

L2 ANSWER 41 OF 64 MEDLINE
AN 1998229889 MEDLINE
DN 98229889 PubMed ID: 9568467
TI Inflammatory bowel disease: no association between allele combinations of the interleukin (IL) I beta and IL-I receptor antagonist gene polymorphisms.
AU Hacker U T; Bidlingmaier C; Gomolka M; Keller E; Eigler A; Hartmann G; Folwaczny C; Fricke H; Albert E; Loeschke K; Endres S
CS Medizinische Klinik, Klinikum Innenstadt, University of Munich, Germany.
SO EUROPEAN JOURNAL OF CLINICAL INVESTIGATION, (1998 Mar) 28 (3) 214-9. Journal code: 0245331. ISSN: 0014-2972.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199806
ED Entered STN: 19980625
Last Updated on STN: 19980625
Entered Medline: 19980615
AB BACKGROUND: **Interleukin 1** (IL-1) and its physiological antagonist **interleukin-1** receptor antagonist (IL-1 ra) play a crucial role in the pathogenesis of **inflammatory bowel disease**. **Polymorphisms** in the genes coding for these cytokines, the restriction enzyme TaqI **polymorphism** for IL-1 beta and the variable number of tandem repeats (VNTR) **polymorphism** for IL-1 ra, have been shown to influence cytokine synthesis in vitro. Recently, an association has been described for distinct allele combinations of these two **polymorphisms** in patients with **ulcerative** colitis and with Crohn's disease but not in healthy control subjects. METHODS: We studied 56 patients with **ulcerative** colitis, 64 patients with Crohn's disease and 196 healthy control subjects. All were unrelated Caucasians of European ancestry. After polymerase chain reaction (PCR) the amplification products were analysed on agarose gels. For the IL-1 beta **polymorphism** the PCR product was additionally digested using the restriction enzyme TaqI. RESULTS: The allele and genotype frequencies as well as the carriage rates of the IL-1 beta TaqI **polymorphism** in healthy control subjects were in agreement with previous findings in other populations. Allele and genotype frequencies of the IL-1 beta **polymorphism** were not different in **inflammatory bowel disease** patients compared with healthy control subjects. Comparing allele combinations of both **polymorphisms** no association could be identified either within healthy control subjects or in the groups of patients with **ulcerative** colitis or Crohn's disease. CONCLUSION: Thus, we could not confirm the results of a previous study reporting an association between the IL-1ra and IL-1 beta gene **polymorphisms** in patients with **inflammatory bowel disease**.

ib,ab 111

L15 ANSWER 111 OF 182 CAPLUS COPYRIGHT 2002 ACS
AN 2000:6195 CAPLUS
DN 132:343849
TI **Improved** direct sequencing system for accurate detection of heterozygosity in HLA-A, B and C loci
AU Bettinotti, M. P.; Mitsuishi, Y.; Lau, M.; Terasaki, P. I.
CS UCLA Tissue Typing Laboratory, Los Angeles, CA, 90095, USA
SO HLA: Genetic Diversity of HLA Functional and Medical Implication, [Proceedings of the International Histocompatibility Workshop and Conference], 12th, Saint-Malo and Paris, France, 1996 (1997), Meeting Date 1996, Volume 2, 373-374. Editor(s): Charron, Dominique. Publisher: EDK, Medical and Scientific International Publisher, Sevres, Fr. CODEN: 68MRA5
DT Conference
LA English
AB A method for direct sequencing of PCR products obtain from genomic DNA was developed that can be used for the typing of HLA-A, B and C loci. The method uses **nested PCR** amplification of genomic DNA and cycle sequencing of the PCR product. The direct and reverse sequence obtained for each locus are compared with a consensus sequence of exon 2 or exon 3 of HLA class I genes. Heterogeneous positions are detd. and the 2 possible sequences are compared to aligned known sequences for the HLA-A, B and C loci to assign the most probable alleles.

L7 ANSWER 601 OF 1437 MEDLINE DUPLICATE 226
AN 97216803 MEDLINE
DN 97216803 PubMed ID: 9062968
TI Development of PCR-SSOP for the identification of HLA-A*02 subtypes and determination of HLA-A*02 frequencies within different ethnic populations.
AU Williams F; Middleton D; Savage D; Gorodezky C; Wilson D W; Fitzgerald J M; Urbaniak S J
CS Northern Ireland Tissue Typing Laboratory, City Hospital, Belfast, Northern Ireland, United Kingdom.
SO TISSUE ANTIGENS, (1997 Feb) 49 (2) 129-33.
Journal code: 0331072. ISSN: 0001-2815.
CY Denmark
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199706
ED Entered STN: 19970709
Last Updated on STN: 19970709
Entered Medline: 19970620
AB A PCR-SSOP typing method, involving a single PCR amplification in conjunction with 19 **digoxigenin** labelled oligonucleotide **probes**, has been developed for the identification of 17 known HLA-A*02 alleles. The method has been applied to four populations (Northern Ireland, Singapore Chinese, Shetland Island and Mexican) and percentages of HLA-A*02 alleles determined within each population.

L17 ANSWER 107 OF 299 MEDLINE
AN 97454802 MEDLINE
DN 97454802 PubMed ID: 9309232
TI [PCR value in the diagnosis of feto-placental human parvovirus B19 hydrops fetalis: apropos of 10 cases].
Valeur de la PCR dans le diagnostic de l'anasarque foeto-placentaire a parvovirus B19: a propos de 10 observations.
AU Wattre P; Thirion V; Bellagra N; Subtil D; Andreoletti L; Hober D; Lion G; Dewilde A
CS Service de virologie du Centre hospitalier universitaire, Institut Gernez-Rieux, Lille.
SO ANNALES DE BIOLOGIE CLINIQUE, (1997 Jul-Aug) 55 (4) 327-31.
Journal code: 2984690R. ISSN: 0003-3898.
CY France
DT Journal; Article; (JOURNAL ARTICLE)
LA French
FS Priority Journals
EM 199710
ED Entered STN: 19971105
Last Updated on STN: 19990129
Entered Medline: 19971020
AB Human parvovirus B19 primary infection during pregnancy is responsible for 27% of non autoimmune hydrops fetalis. Parvovirus B19 antigen detection and parvovirus B19 IgM and IgG antibody determination using enzyme immunoassays are not reliable for diagnostic purposes and lack of specificity. Parvovirus B19 DNA detection in amniotic fluid, fetal blood, ascitic fluid, and fetal biopsies or placenta specimens seems to be the best method for the diagnosis. Ninety-seven samples from 70 cases of spontaneous abortions after fetal death or hydrops fetalis were examined using PCR. A 270-bp length fragment of the NSI gene was amplified using PCR followed by electrophoresis, by Dot-blot hybridization assay using a biotinylated **probe** and by Southern-blot hybridization assay using a horseradish peroxidase-**labelled probe** followed by **chemiluminescent** assay. The Southern-blot hybridization assay was the longest test but the most sensitive. The parvovirus B19 genome was identified in 10 cases. In two cases, intrauterine blood transfusions led to the cessation of symptoms and to the birth of normal babies.

L15 ANSWER 88 OF 182 MEDLINE DUPLICATE 42
AN 1998321908 MEDLINE
DN 98321908 PubMed ID: 9660471
TI Detection of the filarial parasite Mansonella streptocerca in skin biopsies by a nested polymerase chain reaction-based assay.
AU Fischer P; Buttner D W; Bamuhiiga J; Williams S A
CS Department of Biological Sciences, Smith College, Northampton, Massachusetts 01063, USA.
SO AMERICAN JOURNAL OF TROPICAL MEDICINE AND HYGIENE, (1998 Jun) 58 (6) 816-20.
Journal code: 0370507. ISSN: 0002-9637.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199807
ED Entered STN: 19980723
Last Updated on STN: 19980723
Entered Medline: 19980716
AB To differentiate the skin-dwelling filariae Mansonella streptocerca and Onchocerca volvulus, a nested polymerase chain reaction (PCR) assay was developed from small amounts of parasite material present in skin biopsies. One nonspecific and one specific pair of primers were used to amplify the 5S rDNA spacer region of M. streptocerca. Biopsies with different microfilaria densities obtained from 104 Ugandans living in an area endemic for M. streptocerca were tested using both the **nested PCR** assay and standard parasitologic assessment of microfilariae. All 82 samples from microfilaria carriers were positive when tested using the **nested PCR** assay. In addition, M. streptocerca DNA could be detected in 16 samples thought to be microfilaria negative. Furthermore, six days following ivermectin treatment, M. streptocerca DNA was found in 12 of 14 microfilaria-negative biopsies. Control skin samples from patients infected with O. volvulus were all negative in the **nested PCR** assay. This assay **improves** the diagnosis of M. streptocerca and will facilitate further epidemiologic studies.

L2 ANSWER 52 OF 64 CAPLUS COPYRIGHT 2002 ACS
AN 1997:750440 CAPLUS
DN 128:60606
TI Relation of **polymorphisms** of lymphotoxin .alpha. and **interleukin 1** receptor antagonist genes to secretion of tumor necrosis factor .alpha., soluble interleukin 2 receptor and **interleukin 6** in Chinese patients with **inflammatory bowel disease**
AU Xia, Bing; Crusius, J. B. A.; Zhang, Guishui; Guo, Haizian; Deng, Changsheng; Meuwissen, S. G. W.; Pena, A. S.
CS Second Affiliated Hospital, Hubei Medical University, Wuhan, 430071, Peop. Rep. China
SO Hubei Yike Daxue Xuebao (1997), 18(3), 209-213
CODEN: HYDXFU; ISSN: 1000-243X
PB Hubei Yike Daxue Xuebao Bianjibu
DT Journal
LA Chinese
AB The relation of lymphotoxin .alpha. and **interleukin 1** receptor antagonist genes to the secretion of tumor necrosis factor .alpha., sol. interleukin 2 receptor and **interleukin 6** was studied in Chinese patients with **ulcerative** colitis. Patients and Methods: Twenty-two patients with **inflammatory bowel disease** (20 **ulcerative** colitis and 2 Crohn's disease) and 10 healthy controls were studied. Lymphotoxin .alpha. gene and **interleukin-1** receptor antagonist gene fragments were amplified from genomic DNA by PCR. Tumor necrosis factor .alpha., sol. interleukin 2 receptor, and **interleukin 6** prodn. from peripheral blood mononuclear cells were measured by ELISA's. Results: The genotype 1 and 2 of lymphotoxin .alpha. was slightly higher in **inflammatory bowel disease** than in the healthy control (11/22 vs 1/10, P=0.049) and allele 2 of lymphotoxin .alpha. was related to higher tumor necrosis factor .alpha. prodn. from peripheral blood mononuclear cells on stimulation. There was no assocn. between **inflammatory bowel disease** and **interleukin 1** receptor antagonist gene **polymorphism**. Conclusion: the lymphotoxin .alpha. gene may play a role in relation to secretion of tumor necrosis factor .alpha. in Chinese patients with **inflammatory bowel disease**.

L2 ANSWER 54 OF 64 MEDLINE
AN 97167081 MEDLINE
DN 97167081 PubMed ID: 9014770
TI Cytokine gene polymorphisms in inflammatory bowel disease.
AU Louis E; Satsangi J; Roussomoustakaki M; Parkes M; Fanning G; Welsh K; Jewell D
CS Gastroenterology Unit, Radcliffe Infirmary, Oxford.
SO GUT, (1996 Nov) 39 (5) 705-10.
Journal code: 2985108R. ISSN: 0017-5749.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199702
ED Entered STN: 19970305
Last Updated on STN: 19970305
Entered Medline: 19970219
AB BACKGROUND: Concordance rates in siblings and twins provide strong evidence that genetic susceptibility is important in the pathogenesis of **inflammatory bowel disease**. The number and identity of susceptibility genes is largely uncertain. Cytokine genes are attractive candidate loci. AIMS: To study allelic frequencies of **polymorphisms** of the **interleukin-1** receptor antagonist (IL-1RA) gene and the tumour necrosis factor alpha gene in patients with **inflammatory bowel disease**. SUBJECTS: One hundred and twenty nine North European caucasoid patients with **ulcerative** colitis, 120 patients with Crohn's disease, and 89 healthy controls. METHODS: Genotyping was performed by polymerase chain reaction. A variable number of tandem repeats (VNTR) in the IL-1RA gene and a single base pair **polymorphism** in the **TNF alpha** gene promoter region (TNF-308) were analysed. RESULTS: No significant differences in IL-1RA VNTR allelic frequencies were noted between Crohn's disease (allele 1: 72.6%, allele 2: 24.7%, allele 3: 2.6%), **ulcerative** colitis (72.6%, 24.3%, 3.1%, respectively), and controls (76.9%, 20.8% and 2.3%). Some 42.4% of patients with **ulcerative** colitis and 43.4% patients with Crohn's disease were carriers of allele 2, compared with 34.8% healthy subjects. The TNF2 allele was modestly reduced in Crohn's disease (13.2%), compared with healthy subjects (21.3%; p = 0.04), and **ulcerative** colitis (21.6%). CONCLUSIONS: The associations demonstrated are modest: these **polymorphisms** are unlikely to be important determinants of overall disease susceptibility.

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     (FILE 'HOME' ENTERED AT 08:55:56 ON 06 JAN 2003)

     FILE 'MEDLINE, CAPLUS' ENTERED AT 08:56:28 ON 06 JAN 2003
L1            0 S "GENETIC FACTORS IN ANIMAL MODELS OF INTESTINAL"
                E SARTOR/IN
                E SARTOR/AU
L2          177 S E96 OR E97 OR E98
L3          183 S L2 OR E63
L4          116 DUP REM L3 (67 DUPLICATES REMOVED)
L5           66 S L4 AND "INFLAMMATION"
L6           45 S L5 AND INTESTINAL
L7           38 S L6 AND ANIMAL
L8            6 S L7 AND FACTORS
                E CANADIAN JOURNAL OF GASTROENTEROLOGY/SO
L9       154184 S E2
L10           0 S L9 AND L4
L11           0 S L4 AND "INTESTINAL MODELS"
L12          50 S L4 NOT L5
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ABSTRACT

L15 ANSWER 110 OF 182 CAPLUS COPYRIGHT 2002 ACS
AN 1997:714085 CAPLUS
DN 128:10723
TI Diagnosis of Plasmodium malariae infection by the polymerase chain reaction
AU Tahar, Rachida; Ringwald, Pascal; Basco, Leonardo K.
CS Centre de Genetique Moleculaire, CNRS, Gif-sur-Yvette, Fr.
SO Transactions of the Royal Society of Tropical Medicine and Hygiene (1997), 91(4), 410-411
CODEN: TRSTAZ; ISSN: 0035-9203
PB Royal Society of Tropical Medicine and Hygiene
DT Journal
LA English
AB A PCR-based diagnostic method was developed to det. P. malariae using the circumsporozoite protein gene as target. A single 30-cycle amplification was sufficient to detect 0.08-0.8% parasitemia. The sensitivity of the assay was **improved** with secondary **nested PCR**